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THE VISUAL FIELDS IN PATHOLOGIC OCULAR DIAGNOSIS

Harold D. Judd, Opt. D.
Detroit, Mich.

To cover the subject of pathology in the time[1] we have is of course out of the question. It therefore becomes necessary to try and select certain phases of this subject that can be covered to the best advantage in the time we have. With this in mind, I wish to state at the start that you will notice a lack of continuity throughout the whole discourse. I shall not attempt to give in detail either the microscopic or macrosopic anatomy except in one or two instances when it is essential to the understanding of the cases stated.

There seems to be at the present time an inclination on the part of many optometrists to take up the study of the central nervous system and the brain. Fortunately this fits in admirably with a branch of pathology which I desire to impress upon you.

New instruments have been devised and recently greatly advocated for the treatment of muscular imbalance, dissociation of accommodation and convergences, and for many of our ocular discomforts, while resting comfortable on the side, covered with many years' accumulation of dust and cobwebs, is an inexpensive piece of equipment capable of producing information of unquestioned reliability and inestimable value.

The past few years have brought forcibly to my attention some of the reasons for the lack of respect for and feeling of incompetency of our profession, namely, the lack of recognition of certain pathological conditions. When I say recognition of—I refer to the recognition of these conditions in their incipient stages, when there is still time for either medical or surgical intervention, with subsequent sav-

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ing instead of loss of an eye, or its vision. In view of the simplicity of the tests, and equipment necessary to make them, I can not help but feel that the medical profession is justified in many of its condemnations, especially since no effort is made on our part, other than the most superficial examination, to recognize these conditions. Since it is within the province of optometry and is most certainly an obligation of the first magnitude to save vision, it becomes an obligation as a professional man for us to take cognizance of the facts and govern ourselves accordingly.

It is the farthest from my intent to cast any reflections or belittle the importance of that branch of optometric procedure relating tc muscular deficiencies. I, too, am just as deeply interested in relieving asthenopic conditions due to these factors, but I am firmly convinced that they are of secondary importance, as compared to the early recognition of certain pathological conditions which almost invariably lead to blindness or the loss of an eye.

I am neither going to state what these conditions are or the means of identifying them, but will leave that for you to see, as each comes to light in that which follows.

First realize that we are dealing with the only organ in the body which is directly an outgrowth of, and intimately connected with, the brain; the importance of this will be manifest in some of the cases following. Let us take up the relation of these two, the brain and the eye, without going into other than surface anatomy.

The brain has three coverings, all of which are continuous with the optic nerve, and the spinal cord. These coverings are the outer or dura; middle or arachnoid; inner or pia. The dura is separated from the arachnoid by a capillary layer of fluid known as the subdural space. The arachnoid is likewise separated from the pia by the sub-arachnoid space, also a lymph space. A cross section of the optic nerve will show the presence of these spaces, and by means of these lymph spaces, lymph circulates for the nourishment of the organs involved.

The optic nerve and ophthalmic artery occupy the foramen rather snugly; at this point also, it is in close approximation to the sphenoidal sinus, being separated as you will see later by but a very thin, bony wall. The smallness of the foramen and proximity of the sphenoidal sinus are predisposing factors in the easy production of retro-bulbar neuritis.

The immediate anatomical environment of the chiasm is extremely important. (1) It is slightly above the groove of the sphenoid bone, slightly forward and above the sella tursica, in which rests the pituitary body. Directly in the posterior angle of the chiasm is the infundibulum which connects the pituitary body with the brain. Directly over the chiasm is the outer tip of the third ventricle, and on either side of the chiasm lie the internal carotid arteries. In this minute space of about 1½ square inches are many important struc-

tures which frequently become the seat of disease, and the commissure is usually involved along with the others.

The nerves from the eyeball to the primary optic centers have been traced in certain forms of toxic amblyopia, from the primary optic centers to the cortical macular centers has been established but not definitely traced. It has been established also that the relative position of the nerve fibers is the same in the eye and nerve as far as the chiasm, and to some extent, in the cortex. In the macular region, each cone has a direct and individual communication with the primary brain centers. At the macular region, only cones are present and they decrease in numbers until the periphery is reached, when they again become more numerous. The rod and cone layer derives its nourishment from the chorio capillaris of the choroid, while the outer neurone layer is distinctly separate in its blood supply, being nourished by the main retinal branches of the central artery. The greater detour which the branches of the central artery must make in order to reach the extreme temporal region has a bearing upon the early loss of the field in certain pathological conditions.

The papillo-macular bundle of nerve fibers bears three different anatomical positions in the optic nerve and chiasm, at the entrance of the nerve into the eyeball; the papillo-macular bundle occupies the outer quadrant of the nerve, at a point about 17 mm. back of the entrance it forms a round bundle in the center of the optic nerve, while in the chiasm, it is oval in shape with the long axis in the horizontal. These anatomical relations have a bearing on some of the peculiar fields found.

From the optic chiasm visual fibers pass along the optic tracts which are formed of uncrossed fibers of one eye and crossed fibers from the opposite eye, including the papillo-macular bundle, bending around the outside of the cerebral peduncles, and become a part of the mid-brain, and pass on into the primary optic centers; namely, the external geniculate body, anterior corpus quadrigeminum and, pulvinar. The bundle of nerve fibers passing from the primary nerve centers, passes through the extreme end of the internal capsule, and from here spreads out into a fan-shaped formation known as the optic radiation.

The communications from the primary optic centers to the fore, mid, and hind brain, have been quite definitely established. The course, and distribution, of the optic radiation in its relation to the several areas of the posterior part of the hemisphere have distinct localizing value in disease. In the anterior part of the radiation, the close proximity of the motor and sensory paths explains the frequent association of motor and sensory disturbance on the side homolateral to the hemianopsia or contra-lateral to the lesion.

Myer has been able to trace three distinct bundles or paths of fibers in the radiation, dorsal, lateral and ventral. The dorsal and lateral faciculi pass directly to the occipital cortex. The ventral

fibers arch forward forming a loop, which extends as far forward as the anterior end of the temporal horn of the ventricle in the temporal lobe, then turn backward to the anterior part of the inferior calcarine cortex. Temporal lobe lesions may therefore be associated with hemianopic visual disturbances.

From the anterior corpus quadrigeminum nerve fibers pass to the Edinger-Westphal nucleus of the oculomotor nerve. It is by means of these that the reflex arc is completed, and stimulation of the retina by light brings about a reflex contraction of the pupil. The pupillary reflex arc is composed of four sets of fibers (according to R. Bing).

1. From the retina to the anterior corpus quadrigeminum.
2. From the anterior corpus quadrigeminum to Edinger-Westphal oculomotor nerve nucleus.
3. From the Edinger-Westphal oculomoter nerve nucleus to the ciliary ganglion.
4. From the ciliary ganglion to the pupillary sphincter.

In group (2) each quadrigeminal body sends fibers to both Edinger-Westphal nuclei. With this hookup as it were, a ray of light thrown upon the retina of one eye causes the consensual contraction of the pupil of the opposite eye. This much of brain anatomy should be firmly fixed in your minds as it is a very valuable asset in the location of certain brain lesions causing loss or diminution of vision. If the lesion is above the primary optic centers, there will be no interruption of the reflex arc. If the lesion is below these centers there will be hemiopic pupillary activity. This reaction is known as the Wernicke pupillary reaction.

Let us take a simple diagram of the optic tract, in this, you will note that the nasal side of the right eye and the temporal side of the left eye are controlled from the left occipital lobe, while the temporal side of the right eye and the nasal side of the left eye are controlled from the right occipital lobe. With these facts before us, we can visualize the location of lesions causing loss of vision to different sections of the eye.

Disease of the visual pathway in the chiasm or posterior to the chiasm is characterized by changes known as anopsias. They are bilateral changes, rarely monolateral. The nomenclature is: loss of half fields is known as hemianopsias, they are binasal or bitemporal; or altitudinal, if above or below the horizontal plane. When the right or left half of each field is affected, we have homonymous hemianopsias. When the temporal or nasal fields are affected, we have heteronymous hemianopsias. When we have a horizontal instead of a lateral division, they are termed altitudinal hemianopsias superior, or up, or inferior, or down, as the case may be. We also have quadrant hemianopsias, denoted as superior temporal or nasal, or inferior temporal or nasal. I am going to take up in gross detail only

three types of cases at this time, namely: Glaucoma, pituitary and injury cases.

I would like to recall to you the distribution of retinal fibers. You will note that great changes may take place in the peripheral fields without damage to the central, pericentral or macular areas. In glaucoma, evidence points to changes in the peripheral fields prior to any great amount of actual ocular discomfort or pain to the patient. Glaucoma in its early and incipient stages, yields to medication or minor surgical intervention in the form of iridectomy or trephine as the case indicates. Later stages sometimes yield to surgical treatment, but with loss of vision of varying degrees, due to atrophy, hemorraghic tendencies, either sub-choroidal, retinal or uvular, or in many cases, vitreous changes or lens changes, or eventually cataract in some cases. The importance of early recognition can not be too strongly stressed, since successful treatment depends so much on it.

Referring back to the beginning, we found that the coverings of the brain were continuous with the optic nerve. With this fact firmly in mind, papiloedema takes on a new signifiicance with us, and a new importance with us, namely: intra-cranial pressure of some type.

The optic nerve involvements—both intra-cranial and intra-ocular (papillitis) manifest themselves in changes of the relative size of the blind spot of Mariotte. Any relative enlargement or indistinct enlargement of the blind spot indicates a pathological change. The direction of the enlargement is also of importance. In toxic amblyopia or myopia of high degree, the enlargement is toward the point of fixation. In glaucoma, the enlargement is from above and below. Sinus infections yield a more or less concentric enlargement of the temporal borders. Any enlargement of two degrees is considered pathological.

Referring back again to the anatomical environment of the chiasm in relation to the pituitary body, you will recall that the chiasm is located above and slightly forward from the pituitary body. The pituitary body is located in a small sunken cavity in the skull, called the sella tursica (meaning Mexican saddle), owing to its likeness to it. For some unknown reason, the pituitary body is predisposed to the development of tumors either benign or malignant. The tumors cause an increase in the size of the pituitary body, which owing to its anatomical environment must of necessity increase in size upward, with consequent pressure in the chiasm. This pressure on the chiasm produces various types of hemianopsias.

There are such a variety of field changes possible and manifest by different types of intra-ocular disturbance that it is impossible to cover them in a discourse of this kind. Perimetrically, there are two general symptoms aiding us in the differentiation of choroid and retinal diseases. Shrinkage of or loss of the blue fields, out of

proportion to the red and green is characteristic of choroidal disturbance, and disturbance of the rod and cone layers of the retina. Contraction of the red and green, with fairly normal blue and form fields is characteristic of changes in the inner layers of the retina and the ganglionic axis cylinders which form the optic nerve.

In all types of choroiditis, as a rule, perimetric deviations from the normal will correspond to pathological findings with the ophthalmoscope.

Night blindness is a symptom of retinitis pigmentosa, perimetrically it shows first, a progressive and rapid contraction of form field, usually concentric. Second, a preservation of central vision for form and colors after peripheral vision is extensively lost. Night blindness of true origin shows a quantitative loss of color sense, particularly for blue, and more marked in subdued light. Marked restriction or contraction for blue in good light increasing under subdued light, and contraction to a less extent of red and green under the same conditions is then an indication of night blindness.

Retinitis is primarily an inflammation of the ganglionic cells and their axis cylinders; characteristic of this disease is contraction of the field for form and color; reduction of the blue field is usually commensurate with that for form, but red and green suffer to a greater extent. After retinitis becomes well established the contraction of the color fields may be proportionately equal.

Syphilitic infections are carried by the blood stream and the contraction for form and color fields is evidenced early. When the disease is in the inactive form as it were, the central nervous system is its usual lodging place.

Nephritic retinitis or albuminuric retinitis is a form of disease in which toxins and hypertension are the etiological factors. The perimetric indications are first: contraction of form and color fields, second: scotomas, indistinct, later becoming absolute, and enlargement of the blind spot.

Diabetic retinitis shows contraction of form and color fields but not to so great an extent.

Leukemic retinitis (pernicious anemia) shows large massive hemorrhagic areas. Perimetrically, scotomas with contraction of form and color fields is manifest.

Lesions of the rod and cone layers and choroid yield scotomas, of which the patient is usually conscious (positive). Lesions of the retinal layers and optic nerve usually yield negative scotomas or those which the patient is not conscious of.

Nyctalopia is a symptom of retinitis pigmentosa. Idiopathic nyctalopia is purely a functional disturbance and shows no changes in the field. True nyctalopias show the typical quantitative loss of color sense, particularly for blue in daylight and particularly more marked in subdued light.

In diseases of the conducting paths and centers, red and green usually show changes first. In hysteria, we have telescopic fields, or small round visual areas for white, while green fields are larger than normal red, causing reversal of the two or interlacing.

Interlacing fields: blue and red or blue and green usually indicates trouble organic in origin, such as chronic interstitial nephritis in which the blue field is within the red or even the green.

Concentric contraction indicates uncomplicated primary optic atrophy, the color fields are usually reduced out of proportion. Red fields are changed first in optic neuritis, blue in chorio-retinitis. Primary optic atrophy, the red and green, may reduce before the concentric contraction of the whole field takes place. The concentric element, however, is the primary and chief characteristic in these cases. Where we have a concentric contraction with irregular areas, it indicates neuritis, and since the nerve and blood supply to the nasal side of the retina is the shortest, changes are manifest first in this field.

We have conditions of heteronymous hemianopsis when the fields are divided in such a manner that both macula are preserved along with the field above and below. Such fields when the colors fall short of the dividing line for form give evidence that the diseased part of the brain is not limited to one hemisphere but that the other hemisphere is involved either by extension or by pressure.

So far our changes have been principally perimetric, but the usefulness of the perimeter is not confined to the taking of fields alone, but is extended to the study of binocular fixation, the study of the fields of strabismus, and to study and chart diplopias.

For the study of the central and pericentral scotomata and blind spot changes as well as toxic amblyopias, the perimeter must give way to the stereo-campimeter. Toxic amblyopias and lesions of the chiasm and cerebrum are contrasted from chorio-retinal disturbances campimetrically by the sharper outline of the scotoma and the connected or coeval blind spot enlargement.

Toxic amblyopias usually met with are those due to alcohol, tobacco, wood alcohol, arsenic preparations, quinine, coffee, and mercury. The characteristic effect upon the visual field of nearly all of this group is an early loss of vision due to the central scotoma with no pronounced effect upon the peripheral field. The blind spot of Mariotte begins to enlarge with the enlargement more pronounced on the side nearest the macula. Not long after this there appears a small central scotoma, and if the cause is not removed both defects increase in size and finally blend in the form of a long oval with the blind spot at one end and the macula at the other. The defect is always a defect for green first, but usually this stage has passed before the patient reaches our office. Next red is affected and finally white.

Alcohol and tobacco cases are slower in development than syphilitic.

Wood alcohol cases that are truly wood alcohol poison are very rapid in developing and do not improve like other types.

When there is a progress toward improvement the reverse of the onset manifests and the scotoma gradually assumes the shape of a dumbbell, the reduction beginning at the center and this portion disappearing first, leaving one small scotoma at the macula and one at the blind spot.

In arsenic compound scotomas we have first our defect for green, red, then white owing to the pathology of these cases (luetic), the scotoma are hard to outline, poor vision and pain making it impossible at times.

Quinine compounds give marked peripheral changes, restricted white fields with central defect for red. A comparison of the red and white fields is favorable.

Coffee toxicity shows contracted white fields with relative central scotoma, not as large as those of the preceding.

In glaucoma, well, ahead of the usual symptoms, campimetry shows changes in the pericentral area, all glaucoma central defects are connected sooner or later with the blind spot. There are three types of enlargement of the blind spot in glaucoma, (1) a slender prolongation from the blind spot, running up or down or both, known as sickel scotoma, or Seidel's sign. (2) A finger-like process reaching from the upper, lower, or both sides of the blind spot to the fixing area. (3) The Roenne nasal step, which is a step-like process usually in the inferior nasal field, but the superior nasal may also be affected.

Nearly all of the pathological conditions have their peculiar idiosyncrasies and characteristic changes. The chorio-retinal conditions differ from the toxic, nerve tract, chiasm, and brain lesions, by a soft and gradually fading outline, and non-participation of the blind spot.

Going back over this discourse in part will bring to your mind these facts first. Beginning restriction of fields or enlargement of the blind spot is indication of pathological changes, second: hemianopsias, an indication of intra-cranial complications. Since these are both successfully treated, only upon early recognition and since it is within, both the province, and power, of the optometrist to recognize these changes, it becomes an obligation of the first magnitude, if he be a professional man that he be not so shiftless as to pass them by.

I am not belittling the importance of any procedure on the part of the optometrist to give relief to asthenopias due to muscular disturbances or any other cause he may feel is within his province, but they fall into insignificance when placed beside the saving of eyesight and eyes, especially when the time consumed is so insignificant as compared to the technique of muscular calesthenics.

As before stated the animosity on the part of the ophthalmologists in the past has been based principally on the non-recognition of

pathological changes causing him to feel the optometrist incompetent, looking at the situation fairly and with malice to none, he has been justified in many cases. He seldom now refers to our incompetence in other than this field, and frequently admits our equal in refractive ability. Our old alibi of the poor ophthalmologists and their failings in whatever line we chose to see their shortcomings, is but a poor fence for us to hide behind to show our own shortcomings, and is no reason either logical or illogical for our not progressing to a higher standard in our own profession. The fact of poor marksmanship on the part of an adversary is no reason for our placing ourselves as an open target for his remarks which in the field mentioned are correct in the greatest majority of cases. Put the other fellow out of your sight and mind in so far as his shortcomings in your estimation is concerned and be a bigger man than he by minding your own business to the point of becoming his superior in the field you have chosen and his equal in part of his own.

I have a very fine foundation for these facts, having been associated with the ophthalmologists for a period of over twenty years. I have found them on the whole to be a very fair-minded group and fair in their judgment with the men in our profession who were really trying and producing. I am referring to the ophthalmologists, not the near, or general practitioner with a trial case. I am not biased in any way in my personal feelings in this matter and am simply placing it before you as it exists, in hopes that you may see the light and strengthen the weak spots in your foundation, and that in so doing we may have the deepest respect from our nearest colleagues, the ophthalmologists.

DR. HAROLD D. JUDD,
DAVID WHITNEY BLDG.
DETROIT, MICH.

FAULTY METABOLISM NOT A FACTOR IN THE FORMATION OF CATARACT

W. H. Glazer, Ll. B., Opt. D.
Philadelphia, Penn.

I shall herein review the details of the premises on which I held[1] that the crystalline structure is not supported by the usual or by any process of metabolism, as we commonly understand the term.

That a part of the body can exist in a wholesome state unnourished is evidenced, I think, in the external part of a tooth. It is living tissue—tissue consisting of living cells capable of performing acts of ingestion, digestion and excretion—only, that can be said to support the processes of metabolism.

Physiologists frankly admit they do not understand the processes of nutrition of the lens. It can hardly be said that they had not means of detecting the presence of lymph or excreta. It would have been incredible that a search for these had not been made, and the actual course of flow thereby determined.

Anatomists are pretty well agreed that the lens is unorganized, having neither vessels nor nerves. Failure of vessels of course is unimportant; but, failure of nerves and nucleated cells appears to me an insurmountable barrier to any theory of metabolism of the crystalline that could prove reasonably sound.

Now, therefore, any theory that shall explain the creation of cataract, will have to avoid the meaningless (to me) phrase, "interference with the processes of nutrition."

My own thoughts on the matter, which in no way breaks down

[1Dr. John C. Neill, optometrist of Philadelphia, writes: "At the June meeting of the Philadelphia Academy of Optometry the program consisted of a debate between Dr. Wm. Glazer and myself. The subject debated was, 'Is Faulty Metabolism the Cause of Cataract?' Dr. Glazer took the negative and I the affirmative. The debate was judged by Dr. Maxwell Herman, M. D., who is Professor of Ocular Pathology at the Pennsylvania State School of Optometry." The above is a review of Dr. Glazer's remarks. A summary of Dr. Neill's portion of the debate appears on page 499 of this issue of the American Journal of Optometry.]—EDITOR, THE AMERICAN JOURNAL OF OPTOMETRY.

previous views advanced, are that cataract has its prime inception in any force or forces that will cause dehydration or prevent rehydration of a part or the whole, after normal dehydration. It seems reasonable that a normal amount of dehydration takes place in ordinary processes of accommodation and relaxation; that there is, from time of birth, loss of an infinitely small quantity of fluid and its containing saccules, perhaps explaining the normal changes of amplitude of accommodation. It seems more easy to visualize the receding of the far point due to change in bulk and curves than to a change of index of a substance not easily alterable.

No doubt there are many agencies whereby dehydration might be accelerated. Accepting Tscherning's theory of accommodation as being most probably correct, unusual accommodative stresses, as tonic spasms; rises in intra-ocular temperature due to changes in pressure, or the humors conduiting excessive blood heats or heats due to excessive radiant energy, or the cracking of the water molecule by some vibration of a particular frequency, could dehydrate; dehydration through incision is a natural consequence; perhaps other agencies suggest themselves to you.

Returning to the idea universally accepted that cataract is due to faulty metabolism, it would appear that a dead eye ought without much delay go into the cataractous state; yet, I have observed the lenses of ten animal eyes that had been kept in an airtight glass container for more than a week to be beautifully transparent and intact. True, the capsule itself gave off a putrefactive odor; but the lens proper gave off nothing but an agreeably pleasant, faint, drying seaweed tang. (The putrefactive odor of the capsule disturbs my notions somewhat.) Does the capsule have a nucleated cellular structure? Authorities do not expressly say. Would be desirable as a pressure (perhaps, also, temperature) evener.

It is reasonable to ask, if the dehydration theory be a fact, when, then, is the opaque curtain. I can only guess at the answer; friction after dehydration of the containing elements; charring of the elements chemically or mechanically, or normal opaqueness of the containing saccules, suggest themselves.

I may be the guilty possessor of a highly imaginative mind, capable of visualizing distortedly, yet I present these thoughts in the hope that a link of truth may be forged or thought on an important problem be stimulated.

If the thing were feasible, I should like some experimentalist try to rehydrate, not directly, but by introducing and diffusing hydrogen and oxygen gases. The penetrability of the gases into the collapsed saccules would seem to be the only effective means of rehydrating.

DR. W. H. GLAZER
1508 W. SUSQUEHANNA AVE.
PHILADELPHIA, PENN.

FAULTY METABOLISM A FACTOR IN THE FORMATION OF CATARACT

John C. Neill, Opt. D.
Philadelphia, Penn.

In taking the affirmative side of this subject,[1] I have referred for my material to the works of authors whose viewpoints are the result of actual biophysical research and in practically every instance this research has been done in the past year or two. So that we may all have a better understanding I think that it would be well at this time to take a few minutes to explain just what is meant by metabolism. Metabolism is a series of chemical changes, some of which are related to the food material supplied to the molecules of living tissue while others are related to molecules of the living tissue itself. Of these changes occurring within the molecules some are destructive in character whereby the molecule is in part eventually reduced through a series of chemic changes to simpler compounds which are eliminated from the cell, these chemic changes are the result of functional activity and the process is known as katabolism. Anabolism might well be said to be a series of ascending chemic changes whereby part of the food material supplied by the blood plasma is changed to lining material thereby repairing it and restoring it to its former physiologic condition.

So that we may have a better understanding of the pathological process of cataract formation I shall quote from the recent work of Duke-Elder who reviews the recent additions which have been made to our conceptions of the metabolism of the normal lens. The essential reaction in all metabolic processes is a reciprocal process of oxidation and reduction, which throughout the body generally is in large part carried on by the respiratory gases mediated by the blood stream. In addition, however, to this external respiratory mechanism, there is an internal mechanism carried on by substances in the cells themselves which possess the property of spontaneous oxidation.

[1Refer to footnote on page 497 of this issue of the American Journal of Optometry.]—EDITOR, AMERICAN JOURNAL OF OPTOMETRY.

The self-oxidizable constituent of living cells is called glutathione, and it changes from a reduced form as it occurs in the tissues to an oxidized one, the change involving the dehydrogenation of sulphur. There are present also substances known as thermo-stable residues which are capable of actively reducing the glutathione to its original state after it has been oxadized, a combination of the two reactions thus gives a balanced autoxidation mechanism maintained in a constant chemical equilibrium. The lens is an avascular structure, and this internal system, which elsewhere in the body is of secondary importance, presumably becomes the central fact in metabolism, and if it were destroyed or partially put out of gear it would seem to follow that the transparency of the lens must suffer.

While I had intended to eliminate from this discussion as much biochemistry as possible, I find that for a proper handling of the subject it is necessary to inject quite a little. It has long been suspected by investigators that the precipitation and coagulation of the lens proteins was the cause of cataract. The lens proteins are four in number, albumoid a water insoluble protein found in the nucleus; two water soluble crystallines, alpha and beta found in the cortex; and albumin in small quantity. The active constituents of the aforementioned internal autoxidation system are found in large amount in the beta crystalline and in less amount in the alpha crystalline, while the albumoid contains practically none. It is significant that with age the insoluble albumoid increases and the soluble crystallines decrease, a process which is thus accompanied by a gradual and parallel diminution of the reactive elements, while in cataract the same changes occur.

The formation of an organic opacity in the lens is essentially a coagulation of the proteins of its fibres. Recent work has shown that the coagulation of protein is a complex process, which takes place in two stages, denaturation and agglutination. Some of the factors which influence the occurrence of these processes are, any form of radiant energy—heat, visible light, ultra violet light, and radium and also by energy imparted to the molecules of the protein by a mechanical shock or an undue strain. Correspondingly cataract has been produced experimentally by infra-red rays and visible rays. While coagulation by exposure to ultra-violet requires intense exposures, after lesser exposures the lability of the proteins is increased so that they are readily coagulated by other influences such as changes in the salt concentration of the body fluids while in addition after radiation the efficiency of the autoxidation system is lessened.

The trend of recent research therefore seems to be towards the opinion that the coagulation of lens proteins recognized clinically as cataract is to be associated with the same factors which cause the coagulation of protein in the test tube. Two processes are apparently implicated, first, the effect of radiant energy of any wave length, acting upon the lens fibres, denaturing their proteins, either thermally or abiotically and thus increasing the lability of their colloid system

and rendering them more prone to coagulation by other influences. To the effect of radiant energy on the lens fibres is to be added its action, as well as other factors metabolic or traumatic, in increasing the permeability of the capsule and allowing the more ready occurrence of the abnormal passage of fluid or electrolytes. Second, a derangement of the autoxidation system of the lens, caused either by the direct action of radiant energy upon the lens itself, decreasing its glutathione content or upon the capsule whose efficiency is lowered, thus allowing this substance to escape, or by alterations in the concentration of hydrogen ions and salts induced by the factors already mentioned.

While much of the aforementioned work referred to by Duke-Elder requires corroboration in the sense that it will have to be reduced to known laws of chemistry and physics, a great deal of credit should be given those men who have given us this much and who will be able, in all probability, in a very short space of time, to definitely prove that perverted metabolism is the direct cause of cataract.

DR. JOHN C. NEILL,
4936 NO. BROAD ST.
PHILADELPHIA, PENN.

ASTIGMATISM

Philip Jackman, D. O. S., Opt. D.
Union City, New Jersey

Astigmatism is that refractive condition of the eye in which there is a difference in the degree of the refraction in different meridians so that each will focus parallel rays at different points.

Astigmatism is that condition of an eye in which there are two principal meridians of greatest and least ametropia, each having a different focal plane.

In astigmatism, since the refractive surfaces are not spherical, rays from a luminous point are brought to a focus at different points; the shape of the image may be a line, an oval or a circle, but never a point.

Astigmatism which depends upon irregularity of the cornea and if of high degree may be a part of a general malformation of the face, and it is usually regarded as congenital, and in many cases as hereditary.

When the curvatures of the cornea are increased or diminished, it can either be one or both meridians. When astigmatism occurs after the removal of the crystalline lens it is usually due to a change in the curvature of the cornea from the union of the wound, but it may depend upon the fact that the lens while in position was still itself astigmatic, but the opposite of that existing on the cornea so that the latter was neutralized by the former.

Brown[1] made the above statement and he further states "that in another class of cases the astigmatism which has existed prior to the formation of the cataract disappeared, after the extraction of the crystalline lens which would tend to prove that the defect was located in the latter rather than in the cornea. Astigmatism may also be caused by intra-ocular pressure and by a constant contraction of some of the ocular muscles, as it is sometimes seen in forms of nervous disease."

Fuch[2] claims "that acquired astigmatism may have its cause in the cornea, or in the lens." The former variety occurs when the curvature of the cornea has been altered, either because of diseases

affecting it, or still more frequently because of operations. After every cataract operation and iridectomy a certain degree of corneal astigmatism develops, which indeed diminishes with the consolidation of the cicatrix, but seldom disappears entirely.

Physiological astigmatism is produced by lid pressure. It is temporary and not constant. It does not occur in all eyes and when it is found it is rather the exception.

There are two classes of astigmatism: Regular and Irregular. In the regular type the principal seat of the astigmatism is in the anterior surface of the cornea. It is at this point that the principal change of index occurs. A deformity of one of the internal surfaces of the eye which at the anterior surface would produce considerable astigmatism, has only a slight effect on account of the little difference of the index of the media.

Lenticular astigmatism, generally non-congenital and is usually acquired. It is subdivided into the following classes: Permanent and temporary. Permanent is the type in which the crystalline lens has unrelaxable permanent curvature. Temporary may disappear as a result of ciliary relaxation. Regular astigmatism is further divided into the following classes: (a) Simple. One of the principal meridians are emmetropic and the other at right angles is ametropic. (b) Compound. Both meridians are ametropic, neither focus being on the retina.. (c) Mixed. Both of the principal meridians are ametropic, the focus of one being in front of the retina and the other being behind the retina.

In simple hyperopic astigmatism one meridian is emmetropic, and the other meridian hyperopic. If the focus of parallel rays passing through the vertical meridian is on the retina, then the focus of the horizontal meridian would be behind it. The retinal image is in the form of a horizontal line, and if we made a subjective examination, the horizontal line would appear most distinct in the astigmatic fan.

Simple myopic astigmatism is that condition in which one of the principal meridians focus on the retina and the other meridian will focus in front of the retina, and usually at right angles to the one which focused on the retina.

Compound hyperopic astigmatism is that condition in which both principal meridians are hyperopic and focus behind the retina. Both meridians are hyperopic but of different degrees. As a rule the horizontal meridian is of the greatest degree, and naturally it is furthest from the retina. The retinal image is neither a line nor circle; it is a horizontal oval. This form of astigmatism is the commonest of all classes.

Compound myopic astigmatism is that condition where both principal meridians are myopic and focus in front of the retina. Both meridians are myopic but of different degrees. As a rule the vertical meridian is more defective, and the retinal image is neither a line nor

circle but a vertical oval and in the subjective examination the vertical lines will appear more distinct.

Mixed astigmatism may be due to either one of the following combinations: (a) Simple hyperopic astigma with simple myopic astigma, or, (b) Hyperopia with simple myopic astigma, or, (c) Myopia with simple hyperopic astigma. In any case the retina lies between the foci of the two principal meridians; one being before it and the other behind it. The retinal image is never a line but may be a circle or an oval and neither vertical nor horizontal lines will appear very distinct.

Irregular astigmatism is that variety in which there is not only a difference in the refraction in different meridians, but also in different parts of the same meridian. Irregular astigmatism is further divided into the following: (a) Irregular corneal astigma. This being generally the result of wounds or ulcers of the cornea. It may be congenital or acquired. A good way to determine this is by Placido's disc; the rings will be seen reflected from the cornea as distorted circles. Any curvature of the cornea other than spherical or torodial such as hyperbolic or parabolic ellipitial or conical will produce a form of irregular astigmatism. The peripheral portion of the cornea is normally irregular.

There is no correction by ordinary methods of irregular astigmatism, although sight may be considerably improved by a sphere or cylinder. Laurance[3] says "that it may also be aided by stenopaic spectacles in the form of slits." The theoretical remedy would be a liquid kept in contact with the cornea by means of a spherical shell, irregular astigmatism (corneal) disappears when the open eye is immersed in water.

Irregular lenticular astigmatism is caused by the adhesions of the iris producing irregularities of the lens capsule. Or in some cases it may be due to an oblique position of the lens as a result of traumatic causes. Other forms of astigmatism are as follows:

Symmetric astigmatism is present when the combined values in degrees of the meridians of shortest and (or) longest radi of curvature in both eyes equal 180.

Asymmetric astigmatism is present when the direction of the axis of one cylinder bears no relation to the direction of the axis of the other or their combined values do not equal 180.

Homonymous astigmatism is present when the axes of the cylinders are the same in each eye.

Heteronymous astigmatism is a condition in which the defect in one eye is with the rule, and in the other eye it is against the rule. Astigmatism against the rule is a condition in which the horizontal meridian of the cornea has the shortest radius of curvature and the greater power, the vertical meridian has the largest radius of curvature and the lesser power. Astigmatism with the rule is a condition in which the vertical meridian of the cornea has the shortest radius

of curvature and the greatest power, the horizontal meridian has the longest radius of curvature and the shortest power.

Spasm of accommodation may occur in any form of ametropia. Furthermore, it may also occur in emmetropia and when it does it is transformed into apparent myopia, but it is mostly found in hyperopia and in hyperopic astigmatism in either simple or compound. In low degrees of hyperopic astigmatism vision is good and frequently quite normal and corrected vision is good. In high degrees vision is subnormal and often remains so when corrected, although as in absolute hyperopia the use of correct lenses improve it. In myopic astigmatism vision must be subnormal, but not to so great an extent as in myopia of similar dioptreal value. Corrected vision is good.

There are no positive symptoms by which the presence of astigmatism can be determined simply by the patient informing you that of the history. In young people, especially if the astigmatism is of low degrees, very few symptoms manifest themselves. The acuteness of vision may be up to the standard and no great difficulty may be encountered in the use of the eyes. Usually, however, vision is impaired. In hyperopia the distant vision remains good but close vision suffers most. In myopia the distant vision suffers most, while close vision is good. However, in astigmatism both far and near vision are affected.

In myopic astigmatism the patient gives evidence of being nearsighted. Laurance[4] says "that even in hyperopic astigmatism the book or reading matter is very often brought closer to the eyes in order to increase the size of the visual angle and thus compensate for the indistinctness of the retinal image."

Astigmatic people often get into the habit of contracting their lids into a stenopaic apparatus in their effort to overcome their defect, and usually all people having high degrees of astigmatism will tilt their heads in order to secure better vision.

Asthenopia is one of the most important symptoms of astigmatism and is manifested in the form of headache. Authorities claim that more than fifty per cent of functional headaches are caused by the astigmatic error of refraction, even where the error of refraction is so slight that vision is not impaired to any extent. A well known author claims "when vision is normal and headache follows the use of the eyes, astigmatism of low degree is indicated and the less the degree the more the pain." Some authorities claim that nervousness, irritability and general discontent are indirect symptoms of astigmatism.

The five chief diagnostic instruments used in the detection of astigmatism are the ophthalmometer, the retinoscope, the stenopaic slit, Placido's disc and the astigmatic fan used with the subjective test.

The object of the ophthalmometer is the measurement of corneal curves by means of catoptric images viewed through a telescope.

The construction of the ophthalmometer contains a telescope whereby an arrangement is found to double the images seen through it. The telescope contains an eye piece, two objectives and a bi-refracting prism made of crystal quartz and is really two prisms. The base of one prism is placed over the apex of the other prism. This prism is adjusted to produce 3 mm. on separation when the telescope is properly focused. The graduated arc which carries the mires is concentric with the cornea. It is graduated into equal spaces and numbered. Each of these divisions represent one diopter. The ophthalmometer is very valuable in detecting incipient cataract. One must always bear in mind the following rules: As a rule there is always a small amount of lenticular astigmatism present and against the rule, and a small amount of corneal astigmatism with the rule. These conditions often neutralize each other. Another rule which should be carefully followed is: Deduct one-half diopter from the readings when with the rule, and add one-half diopter when against the rule.

The retinoscope is used as another objective method of detecting astigmatism. In astigmatism of high error as a rule the optometrist will encounter a band or streak of bright illumination. In order for the optometrist to determine the nature and amount of astigmatism present he must denote the difference between the shadow in one meridian and then the other meridian, **i. e.,** place a plus two lens for your working distance if you work at the distance of twenty inches. If the shadow moves with the light you have a condition of hyperopia. Insert low powered plus lenses until you have a neutral motion in that meridian. Then you proceed to determine the other meridian. If the shadow still moves with, insert weak plus cylinders with the axis placed in the position of the first meridian. Keep inserting plus cylinders until you have a neutral motion. Another method is by using spheres only. The weakest lens which is found to give a neutral motion will be the spheres and the other lens which is determined in the opposite meridian and the difference between the two will be the cylinder that must be combined with the sphere.

The purpose of the stenopaic slit is to cut off all rays of light at right angles to its position in front of the eye. When placed at the vertical axis all rays in the horizontal meridian are excluded, and when placed at the horizontal axis all rays in the vertical meridian are excluded.

The Placido's Disc is very useful to determine whether there is actually any great amount of corneal astigmatism present. It consists of a disc of cardboard, one surface of which bears a number of black concentric rings upon a white background. An aperture in the center of the disc, and corresponding to the center of the rings, allows the examiner to look through the disc.

The disc is held so that the side with the rings upon it is turned toward the eye that is being examined, and so the plane of the disc is parallel to the base of the cornea. If now we look through the central aperture at the eye we see the rings mirrored upon its cornea.

If the cornea has the normal curvature, these rings will appear perfectly circular. If the cornea has abnormal curvature, the rings are transformed into ellipses, or show irregular bulgings, according as regular or irregular astigmatism is present.

The subjective method consists of cylindrical lenses and an astigmatic chart. The clock dial is the most commonly used. We will assume that we are using the clock dial and that the patient's vision is brought down to at least 20/40, then we proceed to determine if any astigmatism is present. Assuming that the patient is hyperopic, we ask him to inform us which lines are the clearest.[5] If he answers that they all appear uniform, then there is no astigmatism present. On the other hand, should he inform us that the vertical lines are most distinct we immediately know that the horizontal lines are the least clear. We insert weak plus cylinders with the axis over the indistinct lines. In this case we would place the axis at 180°. Keep inserting cylindrical lenses until the patient informs us that all lines look alike to him. As a rule those patients who select the horizontal lines or nearest it are usually hyperopic and those who select the vertical lines as the clearest or nearest it are usually myopic.

In correcting astigmatism for distance the full findings are ordered in myopic astigmatism and in mixed astigmatism. In compound hyperopic astigmatism the spherical lens is usually weakened to meet the requirements of accommodation, but the full power of the cylinder is given. In compound myopic astigmatism the spherical lens is usually reduced for close vision.

DR. PHILIP JACKMAN,
955 BERGENLINE AVE.,
UNION CITY, N. J.

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OCULAR PSYCHOMETRY

Carl F. Shepard, O.D., D.O.S.
and
Wm. Arthur Mendelsohn, Oph. D., Opt. D.
Chicago, Ill.

CHAPTER 12

If the foregoing chapters of this herewith concluding series have to any degree aided any optometrist in his struggle to make his services more valuable, they have accomplished their purpose. No matter how valuable one's services may be, that value will not be appreciated if the service is crudely delivered. No successful jeweler would think of delivering a fine diamond wrapped up in a twist of newspaper; for the same obvious reason, professional services should be delivered through the medium of especially constructed instruments rather than crude, obviously "home-made" devices and awkward make-shifts of equipment intended for entirely different purposes. No optometrist will realize his ambition to enjoy a profitable, professional practice until he has learned to present his services in a professional manner.

It is the purpose of this chapter to be of assistance to those interested in that respect.

First of all, it is necessary to reconnoiter the present optometric situation in order to secure an absolutely unprejudiced point of view.

One must realize that optometry has but recently been recognized as a profession by any one, and is by no means recognized as more than a semi-professional business by many "registered optometrists."

What "optometry" *should be,* is a matter of personal opinion, not now at point. How to make a success of professional practice by those who believe "optometry" should be such, is the problem to the solution of which this effort is dedicated.

We venture the following definitions of professional practice and semi-professional-business to the end that the reader may classify his own opinion of what optometry should be according to the distinc-

tions intended by the authors, and profit by the suggestions made, whether or not personal opinions are in accord.

> A professional practice is that personal service, usually in the solution of human equations and problems, from which the practitioner properly expects *adequate remuneration for his individual skill and ability, without profit from articles of merchandise* involved in such services, and not determined by actual time and labor required, but depending entirely upon the value to the patient of the personal skill and ability of the practitioner in the adjustment of such problems as he professes to be in position to understand.
>
> Semi-professional business is that occupation from which those therein engaged expect just remuneration for individual skill and ability *in the selection and adaptation of merchandise according to the purchaser's actual need,* and additional *remuneration as fair profit on the merchandise delivered.*

There are, of course, some, using the title "optometrist," who merely sell optical merchandise and specialize in filling ophthalmic prescriptions written by others, and who admittedly place no value upon their individual skill and ability in determining the purchaser's needs; but the majority of such merchants prefer the more correct name "optician."

If the reader will now count the "registered optometrists" in his community, then list separately *those who expect adequate remuneration without profit from merchandise,* he will immediately recognize the justice of the layman's common definition of an optometrist—which is:—"one who sells glasses for a living."

As a rule, when the average rational citizen goes to an optometrist, he expects to buy a pair of glasses; and is prepared to resist buying two pair, just as he is prepared to resist being sold socks when he goes to a haberdasher to buy a shirt. Exceptions to this rule exist only because some optometrists, at least, some of the time, present their professional sides so conspicuously that the merchandise side passes unseen, and in so doing, the door of opportunity to the professional practice of optometry is opened.

A real opportunity, because *people do not want glasses.*

People buy automobiles; Easter hats, and dancing shoes because they *want* them; but people buy glasses only when convinced they *must have them* to assure visual comfort and efficiency.

There is a large and ever increasing number of people who realize, chiefly from experience, that visual comfort and efficiency are derived through the individual skill and ability of the optometrist rather than from the trademarks upon the lens and frame. These people seek the man who depends upon individual optometric skill and ability for a livelihood just as naturally as they go to buy automobiles from the man who sells autos for a living.

Logically, discriminating seekers of professional service prefer the wholly professional optometrist to the semi-professional. Many optometrists claim to be entirely professional because they never prescribe lenses unless lenses are needed, and sell nothing but the highest quality merchandise. Here "professional" is confused with "ethical." Every good merchant believes in his wares, and no successful auto salesman would recommend a delivery truck to a prospect who desires to motor for pleasure.

The professionally aspiring optometrist, having arrived at a logical viewpoint, will not waste time berating and condemning the practice from which professional optometry is being evolved; at least, so long as they be ethical. Rather, he will devote his energies and investments primarily to the task of making his individual skill and ability superior to that of his contemporaries whose energies and investments are divided between professional efforts and merchandising; and secondly, to devising ways and means of distinguishing his professional individuality; and finally, to the problem of fees.

Distinguishing one's individual skill and ability, after being possessed of such qualties, is accomplished by substituting personality and equipment for merchandise displays; and first person, personal pronuons for the names of instruments, lenses and frames.

The furnishings of the office suite wherein a professional practice is conducted surely reflect and advertise the personality of the doctor. He will not permit his individuality to be submerged by displays of merchandise in common with displays in show cases and cabinets of optical merchants. Neither will he permit the effect of his own carefully chosen and correctly uttered words and phrases to be drowned by the colorful clatter of advertised slogans, names and catch words. In short, the professional optometrist must realize that his clientele *wants* individual skill and ability efficiently delivered, and he will be both polite and politic enough to avoid, in every possible way, confusing their wants with merchandise, or cheapening his skill and ability with make-shift appliances.

The optometrist who does no dispensing has already made his appearance, but as yet, in many communities it may be impractical for the professional optometrist to depend upon another to fill his prescriptions. Nevertheless, he can keep his individual skill and ability in the foreground of the transaction by announcing to the patient, at the conclusion of his examination: "I am now familiar with the condition of your eyes, and I am quite sure I understand your requirements. I will take your case for a fee of such-and-such, and without further charge supply you with the necessary means of assuring your visual comfort and efficiency." In other words, name a fee for *his professional services* and supply the glasses as a part of that service.

The problem is much simplified when the optometrist has become sufficiently cognizant of calisthenical possibilities to conscien-

tiously insist upon a thorough course of orthoptics and observation, requiring perhaps several visits to his office, in every case; after which glasses, if unavoidable, are obviously incidental to the professional service rendered.

The professional optometrist must realize, and it cannot be overemphasized, that his reputation for skill, ability and integrity is the only reason for his professional existence. He must further realize that his reputation thrives upon success and is injured by failures. He must, therefore, erect every possible safeguard for the protection of his professional reputation.

Foremost among such safeguards is the necessity of procuring equipment which insures the best expression of the doctor's skill in determination, refraction and physiologic correction. Humanity is forever indebted to an optometrist, Dr. Harry L. Fuog, Professor of Phorometry, Los Angeles School of Optometry, for the means of investigating the efficiency of and improving those ocular functions which precede recognition of the object seen. Before his revolutionizing innovation, optometrists and other refractionists began their examinations only after the patient's eye was fixed upon the object of regard without recognition of the fact that modern industrial conditions exact their greatest toll of the functions of monocular and binocular fixation, orientation, excursion and co-ordination. From Dr. Fuog's beginning, optometrists in professional practices have developed means of demonstrating and more cleary explaining conditions to their patient; and a systematic techique for the correction of many previously hopeless conditions.

Moreover, the equipment so selected should be adapted in every particular to the use intended. It is no longer necessary or excusable for an optometrist to employ devices for which he must apologize, or which requires him to perform ridiculous antics and contortions before the eyes he purports to examine. Neither is it necessary for the modern optometrist to exhaust his own patience and physical energies in directing orthoptic calisthenics, nor to depend upon the rare co-operation necessary if "home exercises" succeed.

Present day optometric instrumentation permits the properly equipped optometrist to investigate and to exercise every ocular function without the sacrifice of professional dignity or prestige.

Another important safeguard is to insist upon sufficient opportunity to examine and study each case. The routine, or preliminary examination must be followed by other appointments until the optometrist, through his study of the case is confident of two equally important factors: First, his own understanding of conditions; and second, the patient's wholehearted co-operation.

Once the optometrist accepts a case, he places his professional reputation in the patient's hands to do with as the patient wills; or worse, subject to the patient's thoughtlessness.

This leads to the question of fees. The fee in every case must

be high enough to command the respect of the patient for the optometrist's skill and ability. If the fee named is less than the value of the patient's own time, demanded in co-operation, he will, then or later, seek one whose fees command his respect. On the other hand, the fee named should be within the ability of the patient to pay.

If the patient is able, but unwilling to pay the fee named, it is because he does not sufficiently value either his own visual efficiency or the optometrist's reputation. For either reason, he should not be entrusted with the doctor's reputation, but should be advised to acquaint himself with it, and to return later.

If the patient is willing, but unable to pay the fee named, and is nevertheless permitted to obligate himself beyond his circumstances, the debt may, probably will, become an irritant and cause the patient to hold the doctor's reputation too lightly.

Obviously then, the professional investigation of a given case should be continued through a sufficient number of appointments to permit a thorough understanding by the doctor of his patient's circumstances, environment, condition and requirements; and on the other hand, by the patient, of the doctor's methods and aims.

A proper fee is unquestionably a safeguard to professional reputation. Furthermore, inadequate fees will not permit the doctor to secure proper equipment, and will prevent investments both of time and money in the constant pursuit of knowledge demanded by the rapid advancement of the science of optometry. Finally, no man can give his undivided attention to patients' troubles when he has financial worries sapping a great deal of his energy.

Another safeguard is the avoidance of arousing the patient's expectations. It is much better for the patient to realize more than he anticipated instead of less. The professional optometrist will say something like this to the patient who demands assurance of satisfactory results:

> "I am quite sure I know the right thing to do in your case, and how to do my part. However, I can do no more than my best. Much depends on you, and upon circumstances over which I have no control. If you have any serious doubt of my skill, ability and integrity, I would rather not risk my reputation on your case."

In conclusion of this, the last chapter of the series, the authors wish to say, as the result of investigations made in every corner of the vast land of optometric opportunity that stretches from the sun's morning bath to its evening plunge, and from Canada's expanses to the balmy tropics, that the professional practice of optometry has today within its grasp, an opportunity greater than that offered any other profession, to render a real and valuable service to humanity. And we hope these chapters encourage active minds to thoroughly investigate achivements of the science of optometry, now being further developed in the professional practice of optometry, which enable the alert and conscientious to promise posterity stronger eyes and greater comfort.

IMPORTANCE OF SPEED IN THE OBJECTIVE EXAMINATION

Josph I. Pascal, B.S., M.A., D.O.S.
New York, N. Y.

The length of a complete eye examination naturally varies with the examiner and the type of case. But while speed is advantageous, as a general rule in all tests, it is especially essential in the objective tests, as will be shown presently.

Every examiner nowadays employs both objective and subjective methods for a complete eye examination. It is important, however, to see that one set of tests should not react unfavorably on the other set of tests. Usually the objective tests are made before the subjective tests, and there are many good reasons for doing so. But there is one disadvantage of this order which must not be overlooked and which I want to discuss briefly.

The retinoscope has justly been called a pathfinder. Even when it gives only an approximate idea of the refractive condition of the eye, it is of great service in facilitating and speeding up the subjective part of the examination. But the strong light which bathes the retina during the retinoscopic test inevitably leads to some dulling of its sensitivity. This in turn leads to a lessened and slower response of the retina during the subjective tests. And, unfortunately, it is just at this stage of the examination, when the refinements of the correction are being worked out, that a highly sensitive and quickly responding retina is especially desirable.

With the ophthalmoscope, because of its intenser light, this dulling of the retina by a strong flood of light is still more marked. For this reason, the ophthalmoscope is best used, except in selected cases, after the subjective tests have been completed. But the retinoscope, as a pathfinder, must be used early in the examination and before the subjective tests, in spite of its undesirable secondary effects.

However, the wise retinoscopist, realizing this deadening effect on the retina, seeks to make his retinoscopy tests as speedily as possible. He tries to make his readings from a brief flash and one or two rotations of the mirror. But the obliquity of observation under which retinoscopy is at present done, precludes the possibility of getting any reliable results from a rapid flash and one or two

movements. There is so much aberration, radial astigmatism, coma, etc., in the emergent light, due to the obliquity of observation and the variability of fixation, that relatively much time is required to be able to study the reflex and its behavior. And this difficulty is found with any form of retinoscope.

The photoscope removes this difficulty. For one of the advantages of photoscopic retinoscopy is that it enables the examiner to work very rapidly. As there is practically no obliquity of observation, and no change in fixation, the reflex or emergent light from the patient's eye presents none of the disturbing aberrations otherwise found. Therefore, a flash with the retinoscope and one or two movements tell the examiner in a moment's time the nature of the error and the extent of its neutralization. Full retinal sensitivity is thus retained for the finely discriminative subjective tests.

DR. JOSEPH I. PASCAL,
NEW YORK, N. Y.

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EDITORIAL STAFF

DR. CAREL C. KOCH, Editor
209 Yeates Bldg., Minneapolis, Minn.

DR. JACK I. KURTZ, Associate Editor
209 Yeates Bldg., Minneapolis, Minn.

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A CASE OF TOBACCO-ALCOHOL AMBLYOPIA ACCOMPANIED BY A DIPLOPIA

Patient, male, age 61. Occupation, merchant. Patient first came to me in 1919. At that time I prescribed the following bifocal correction:

O.D. + 0.75 D.Sph. ⊃ + 0.75 D.Cyl. axis 180.
O.S. + 0.50 D.Sph.
Add, O.U. + 1.50 D.Sph.

These were used until 1922, when he again returned and had his bifocal correction changed to:

O.D. + 0.75 D.Sph. ⊃ + 0.75 D.Cyl. axis 180.
O. S. + 0.75 D.Sph.
Add, O. U. + 2.00 D.Sph.

This proved satisfactory until 1925, when he returned. His only complaint being at that time that his eyes seemed to become tired while doing near-point work. The formula was changed to:

O.D. + 1.00 D.Sph. ⊃ + 0.75 D.Cyl. axis 180.
O. S. + 1.00 D.Sph.
Add, O. U. + 2.25 D.Sph.

This gave him perfect comfort until January of this year, when he

again returned saying that he was suffering slightly when doing near-point work. His correction was again changed this time to:

O.D. + 1.25 D.Sph. ⊃ + 0.75 D.Cyl. axis 180.
O. S. + 1.25 D.Sph.
Add, O.U. + 2.50 D.Sph.

This gave him perfect vision. At the time of this last examination, his phorias were, uncorrected at six meters, 1½ degree of esophoria, and corrected ½ degree of esophoria. The same test made at his occupational near-point, 39 cm., indicated 3½ degrees of exophoria. His corrected ductions were as follows:

Ductions at 6 meters.

O.D.	Abd. 6/3.	Add. 15/9.	Super. 1/0.	Infer. 1/0.
O. S.	Abd. 6/4.	Add. 15/9.	Super. 1/0.	Infer. 1/0.

Ductions at 39 cm.

O.D.	Abd. 6/4.	Add. 21/12.	Super. 1/0.	Infer. 1/0.
O. S.	Abd. 6/4.	Add. 21/14.	Super. 1/0.	Infer. 1/0.

This new correction was used with the greatest of comfort until May 3rd when he noted a slight blurring of his distance vision. At this time, he says, he thought little of it, thinking perhaps it might be caused by indigestion or something of that sort which would quickly pass away. On the 5th of the month, however, he was aware of a slight but nevertheless noticeable doubling up of the distance images. The following Monday, the 7th, he called me on the phone, explaining at this time that his vision was not as clear as it had been and that for the past two days everything be looked at at some distance from him appeared to double horizontally. I immediately suggested a complete physical examination which he started on the same day. On the 10th, he came to the office. The physician who had examined him had found nothing wrong and had told him that his eyes must be the cause of the trouble. On the 11th I examined the case without a cycloplegic but could find no abnormal symptoms on viewing the fundus or the media. All reactions were normal and his distance vision could not be improved beyond a poor 20/30. When I had prescribed for him in January it had been, when corrected 20/20, or a bit better. On the 12th his fundus was again examined, by both an ophthalmologist and myself, his pupils being dilated by cocaine. The eye-grounds were apparently normal. On the 14th, a most searching examination was made by the writer, the principle results being to note that the patient's color fields for both red and green were very restricted, the blind spot was somewhat enlarged and that single binocular vision could be had by incorporating 11 prism diopters base-out with his distance correction.

The restriction of the color fields and the enlarged blind spot were indicative of a toxic condition. His history chart showed that he used both alcohol and tobacco in moderation. Upon further questioning it developed that he smoked from 5 to 8 cigars a day, in

addition to chewing tobacco while at work. He also claimed, that while he chewed, he never spit, stating he never felt the need of so doing. He also said that he was unaware of ever swallowing any of the tobacco juices mixed with saliva, though he guessed must have done so, for he was emphatic that at no time while he chewed did he ever spit, though frequently he chewed for two or three hours at a time while at work in his store. This practice he had started more than 40 years before and he had continued it regularly since. As far as alcohol was concerned he claimed he did but little drinking, only some beer which he made himself, from a new, splendid recipe which he had gotten in March of this same year. Further questioning revealed that his consumption of home-made beer would run from 8 to 10 quarts per week. He estimated the alcoholic content of this beer was from six to eight per cent.

The diagnosis was then tentatively made by the writer as a case of toxic amblyopia due to combined tobacco and alcoholic poisoning, although the very apparent diplopia which accompanied the amblyopia could not be accounted for by this diagnosis.

The treatment was as follows. All tobacco, both smoking and chewing, and all forms of alcohol were denied the patient. His bifocal correction was temporarily taken from him and two pairs of glasses were substituted, one for distance and one for near-point work, these corrections being of similar strength as were the various elements of the bifocals he had just been using. This was done as the bifocal element itself seemed to aggravate the diplopia, and also single vision treatment corrections could be more easily changed from time to time as necessity demanded, than could bifocal corrections. The patient was also instructed to take a Turkish bath every other day for the next three weeks, and to get at least nine hours sleep every night. He was also instructed to come to the office daily for treatments. All these instructions were faithfully carried out during the following month.

The office treatments consisted of base-in oculo-prism treatments at six meters in addition to the use of high-frequency. The oculo-prism treatments at first were started with 11 prism diopters base-out before the patient's eyes in addition to his distance correction, the shock or jump technic being employed, the treatment prisms being base-in. At the end of six treatments the patient had single binocular vision with 7 prism diopters base-out before his eyes instead of 11 as was needed just a week before. The high-frequency treatments were administered binocularly for from two to four minutes each day, the current being very weak, of such a strength that the purple rays are just visible in the glass vacuum electrode.

At the end of the first week, his distance treatment correction was changed from the previous sphero-cylindric formula to the following:

O.D. + 1.25 D.Sph. ⌒ + 1.25 D.Cyl. axis 180⌒ 3½$^{\Delta}$ base-out.
O. S. + 1.25 D.Sph. ⌒ 3½$^{\Delta}$ base-out.

This gave him single binocular vision, the treatments being continued as before. At the end of the second week the base-in oculo-prism treatments had so improved his extrinsic muscular condition that he was able to get single binocular vision with 4 prism diopters base-in instead of 7 as needed a week before. The treatment correction was then changed to:

O.D. + 1.25 D.Sph. ⊃ + 1.25 D.Cyl. axis 180⊃ 2$^{\Delta}$ base-out.
O. S. + 1.25 D.Sph. ⊃ 2$^{\Delta}$ base-out.

He also at this time claimed he noticed an improvement in his distance vision, he now being able to read all of the 20/30 chart and some of the characters on the 20/24 foot chart. This treatment correction he used for another week, the office treatments continuing as before, high-frequency and base-in ocular-prism treatments. At the end of the third week he had binocular single vision with 1 prism diopter base-out added to his distance correction instead of a 4 base-out as needed just a week previous. The treatment correction was then changed to:

O.D. + 1.25 D.Sph. ⊃ + 1.25 D.Cyl. axis 180.
O. S. + 1.25 D.Sph. ⊃ 1$^{\Delta}$ base-out.

Treatments as previously outlined were continued for ten more days. At the end of this period his vision had improved to a point where he could read all characters of the 20/20 chart except the figure 8, and he no longer suffered from diplopia. His abduction at six meters at the end of one month of base-in oculo-prism treatment was 7 break, and 5 return.

He was then instructed to continue his abstinence and to report any changes which might take place in his vision. The treatments and the single vision treatment corrections were discontinued, he returning to the bifocal correction previously prescribed in January. On the 1st day of September, the writer phoned the patient, receiving a report of normal vision with no recurrence of the diplopia.

C. C. K.

BOOK NOTICES

APPLIED REFRACTION. Homer E. Smith, M. D. Published by William Wood and Company, New York City. 132 pages. Illustrated. 1928. $2.75.

Subjective refraction, the use of the ophthalmometer, and cross cylinder technic are subjects which are thoroughly dealt with in this new book on ocular refraction. Other phases of examination procedure are referred to but not in the detailed way as are those just mentioned.

In this work by Smith, the author gives his views on the subject as founded upon many years of successful practice. Smith is an ophthalmologist and uses a cycloplegic in nearly all of his cases, yet, as he says, he has no quarrel with those who do not use cycloplegics. The book he has written is not sent forth to "jamb" his ideas down the throats of all refractionists but rather to add to our sum of knowledge of the subject by outlining his experiences with various technics and to give his reactions to the teachings of others. Several chapters dealing with hyperopia, myopia, astigmatism and presbyopia are cleverly written and the whole work is entertaining and well worth the attention of all optometrists.

He strongly advises the use of controlled illumination in the matter of visual test apparatus; he condemns the use of the astigmatic fan and claims that 20/20 as a standard of visual normal is too low. He advises 20/15 as a better normal. The book is filled with pithy paragraphs such as the two which follow: "Part of the other equipment of the old trial case was designed simply to fill the slots and amuse the children; the varicolored tints, the plano-lens and the pair with crossed lines have no discoverable utility"—and further on, "The complicated accessories for testing imbalances of the ocular muscles fail of their purpose because only imbalances of the primary position fall within their province." C. C. K.

THE PRESCRIBING OF SPECTACLES. Archibald Stanley Percival, M.A., M.B., B.C. Camb. Third Edition. Published by William Wood and Company, New York City. 239 pages, 29 illustrations. 1928. $5.00.

The book, "The Prescribing of Spectacles" has been rewritten and a considerable amount of new material, which embodies the latest findings, has been added. To read this book one must dig deeply into

the subject, yet the subject-matter is of such great interest and there are so many new ideas to be found that the reader will find himself well repaid.

Percival handles the subject-matter in this book entirely different from what we in the United States are used to finding in most books on refraction. Upon reading it, one at once concludes that the writer is a very keen observer, a deep thinker and student of optics. He does not fear to express his views and conclusions as he sees them even though they may be in conflict with other supposedly accepted teachings or views.

For instance, in discussing periscopic lenses, of which the author made a special study, he states that the claims that punctual and katalral lenses are superior to others is not a proven fact. The photographs of test types, which are intended to show the advantage gained by these lenses in definition of the peripheral part of the field are not proof of this fact at all. He grants that these lenses are far better than his own (periscopic lenses) for photographing purposes, but he maintains that such pictures are no evidence to show that they are useful for visual purposes. He maintains that "the optical conditions are entirely different. The photographic lens is provided with a *fixed* stop; the perioscopic, with a *movable* stop (the pupil) and so gives us a well defined macular image of the eccentric part of the field under examination when the eye ranges from side to side. The macula is far more sensitive than any photographic plate, even to a thousandth of a millimeter."

The first 144 pages of the book are devoted to subjects intimately connected with applied refraction, the remainder is the "optical section" and deals mainly with optical problems and formulae and their mathematical derivations. Also a number of important tables are to be found in the last pages of the book.

Reading of this book is stimulating as well as instructive and brings a new light on many refraction problems. J. I. K.

ANNOTATIONS AND SOCIETY PROCEEDINGS

The American Journal of Optometry is the official Organ of the American Academy of Optometry, as well as the Organ of the Arkansas, Delaware, Iowa, Maine, Maryland, Michigan, Minnesota, Nebraska, North Dakota, Oklahoma, Pennsylvania, South Dakota, Vermont and West Virginia Optometric Associations.

The following gentlemen have consented to supply items of interest from their respective sections, acting in the capacity of

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COMMITTEES FOR COMING CONGRESS

Announcement has been made by Dr. John J. McNally, president of the Philadelphia Society of Optometrists, of the appointment of Dr. Otto G. Haussmann as general chairman of the committee on arrangements for the thirty-second annual congress of the American Optometric Association, which will be held in Philadelphia next year.

While the Philadelphia society has been in recess during the summer months, the officers, in conjunction with the president of the Pennsylvania Optometric Association, have had frequent conferences at which plans for the coming congress were the subject of consideration.

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PHILADELPHIA SOCIETY OF OPTOMETRISTS

The Board of Regents of the Philadelphia Society of Optometrists, of which Dr. Albert Fitch is chairman, is making arrangements for a banquet to be held on October 18th. Subcommittees have been appointed to look after the various details and all efforts are being directed toward making the affair a good fellowship party for the optometrists of the city and vicinity.

The program, as outlined at present, shows that many novel features will be introduced for the entertainment of the guests and a highly successful affair is predicted.

The board has also announced an extensive lecture course for the fall and winter meetings, which will be resumed on September 25th.

ACADEMY YEAR BOOK

Dr. J. Fred Andreae, optometrist, Lexington Building, Baltimore, Maryland, secretary of the American Academy of Optometry, informs us that a few of the bound Academy Year Books are still available to optometrists who wish to read the eight technical talks which were published. This book was reviewed in the American Journal of Optometry, pages 35-37, No. 1, Vol. 5. Copies of this work can be secured by writing Secretary Andreae. The cost is $5.00.

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GRADUATE CLINIC IN SAN FRANCISCO

Dr. George Mayerle, Jr., optometrist of San Francisco, writes that a week of graduate clinical work, under the direction of Dr. A. M. Skeffington, was held in that city, the week of August 12th to 17th. A large group of optometrists were in attendance, the study being the newer developments of optometric diagnosis. Practitioners from San Francisco, Oakland, Alameda, Haywards, Richmond, Martinez, Dinuba and Santa Cruz were in attendance and assisted in the work on the many patients who presented themselves.

* * * *

KRATOMETER LECTURE IN NEW YORK

Dr. John F. Zug of Pittsburgh, Pennsylvania, lectured before a group of New York optometrists on Friday, August 24th. The subject of his address was, "Some Observations and Results in 200 Kratometric Cases."

Dr. Edwin Wolff writes that much interest was aroused, particularly by the photographs of actual cases of strabismus, and by Doctor Zug's description of the technic used in each case.

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IDAHO OPTOMETRIC ASSOCIATION

Dr. P. A. Simmons, optometrist of Boise, Idaho, secretary of the Idaho State Association of Optometrists, announces with regret, the death of Dr. Edward S. Owen, optometrist of Boise. Doctor Owen was one of the founders of the Idaho association and a member of the state board. His fellow practitioners, says Doctor Simmons, are deeply grieved at his passing, as the profession in Idaho has lot one of its outstanding members.

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AMERICAN ACADEMY OF OPTOMETRY

The annual meeting of the American Academy of Optometry will be held in Chicago, Illinois, on December 2nd, 3rd, and 4th of this year. The sessions will be held at the Morrison Hotel and all fellows of the Academy are expected to be present. The sessions are entirely educational. The program will be announced later.

PENNSYLVANIA OPTOMETRIC ASSOCIATION

Dr. Howard T. Lewis, optometrist of Pittsburgh, Pennsylvania, chairman of the program committee of the Pennsylvania Optometric Association, announces that the annual convention of that society will be held at the William Penn Hotel at Pittsburg on the 1st, 2nd and 3rd of October.

Other committees and their chairmen are as follows: Dr. Edwin T. Meyer, general chairman; Dr. Wm. J. Van Essen, chairman of general arrangements; Dr. Thomas H. Martin, chairman of banquet; Dr. A. M. McConnell, chairman of education; Dr. F. L. Nungesser, chairman of exhibits; Dr. H. W. J. Seltzer, chairman of publicity; Dr. E. A. Nungesser, chairman of registration, and Dr. J. N. Helman, chairman of entertainment.

Dr. Albert Fitch, dean of the Pennsylvania State School of Optometry and Dr. Charles Sheard, of the section of bio-physics of the Mayo Clinic of Rochester, Minnesota, will be the chief speakers.

* * * *

STATEMENT OF OWNERSHIP

Management and Circulation as required by the Act of Congress of August 24th, 1912, of the American Journal of Optometry, published monthly at Minneapolis, Minnesota, for October 1st, 1928.

State of Minnesota, County of Hennepin—ss.

Before me, a Notary Public in and for the State and County aforesaid, personally appeared Dr. Jack I. Kurtz, who having been duly sworn according to law, deposes and says that he is the Business Manager of the American Journal of Optometry and that the following is to the best of his knowledge and belief, a true statement of the ownership and management of the aforesaid publication for the date shown in the above caption, required by the Act of August 24th, 1912, embodied in Section 411, Postal Laws and Regulations, to-wit:

First, that the names and addresses of the Publisher, Editor, and Business Manager are as follows: Publisher, The American Journal of Optometry Publishing Association, 209 Yeates Building, Minneapolis, Minnesota. Editor, Dr. Carel C. Koch, 209 Yeates, Minneapolis, Minnesota. Business Manager, Dr. Jack I. Kurtz, 209 Yeates Building, Minneapolis, Minnesota.

Second, that the owner is the American Journal of Optometry Publishing Association—Dr. Carel C. Koch, President, and Dr. Jack I. Kurtz, Secretary and Treasurer.

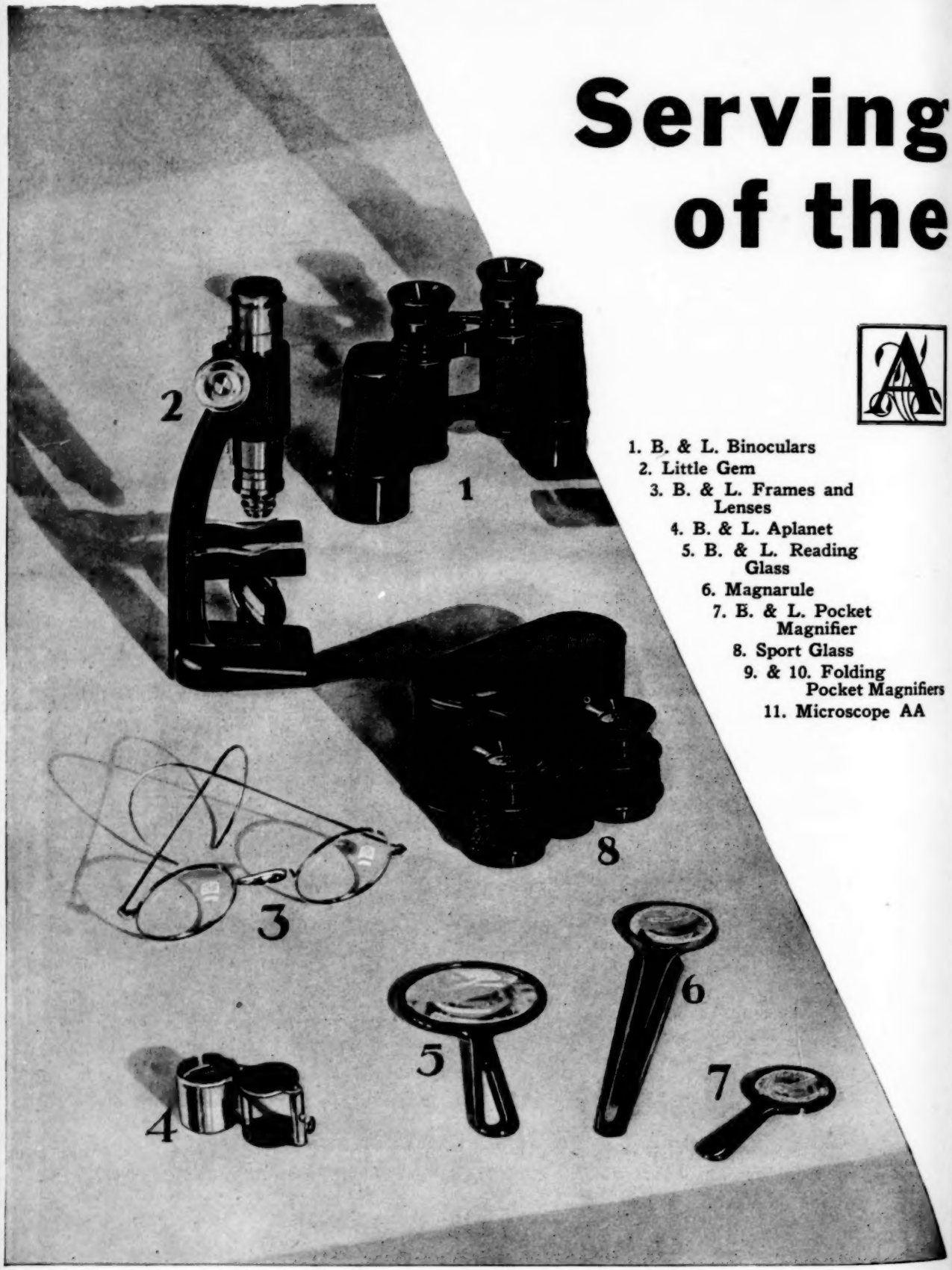
Third, that there are no bondholders, mortgagees, or other security holders owning or holding one or more per cent of the total amount of bonds, mortgages or other securities of this publication.

DR. JACK I. KURTZ, Business Manager,
The American Journal of Optometry.

Sworn and subscribed before me this 28th day of September, 1928.

NEITA S. ROBERTS, Notary Public,
Hennepin County, Minnesota.

(My commission expires Jan. 3, 1931.)



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